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Functional genomics of lung cancer progression reveals mechanism of metastasis suppressor function

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The mechanism of action of NME2, a widely accepted metastasis-suppressor gene, is poorly understood. Recently we found that NME2 directly regulates transcription of the *c-MYC* proto-oncogene. This prompted a genome-wide study to ascertain whether NME2 exerts its anti-metastatic action through transcriptional regulation. Chromatin-immunoprecipitation followed by massively parallel sequencing (ChIPseq) along with transcriptome profiling uncovered a network of genes involved in inter-cellular contact, focal adhesion and actin assembly under direct transcriptional control of NME2. In line with this, NME2-depleted cells displayed increased focal adhesion points and altered actin stress fiber organization. Our findings demonstrate that NME2 regulates transcription of a key focal adhesion factor vinculin and its localization within adhesion foci. NME2-depleted A549 lung cancer cells showed higher invasiveness in vitro and seeded more metastases in vivo. Consistent with these findings, expression of several NME2-transcriptional target genes related closely to advanced tumor stages with metastatic proclivity, and NME2 levels predicted patient survival.

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